Haemodynamic stability of the ventilated intensive care patient: A review

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One of the major reasons for admission to an intensive care unit is haemodynamic instability. It is important for physiotherapists to recognize and predict haemodynamic instability when planning intervention in these patients.

This paper reviews the existing literature on haemodynamic effects of physiotherapy in ventilated intensive care patients.

Recommendations are made for further research, including multi-centre documentation, attainment of normal data and single case studies in this area.

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Physiotherapists are considered to have an important role as part of the intensive care team in order to optimally manage respiratory and circulatory function in critically ill ventilated patients. The Task Force on Guidelines (Critical Care Medicine 1991) states that 24 hour coverage by a respiratory therapist is a minimum requirement for an intensive care unit (ICU).

Physiotherapy is often considered to be a threat to haemodynamic stability (Klein et al 1988) yet despite this supposition, minimal work has been done on documenting events, completing controlled trials or investigating the effect of respiratory physiotherapy on various cardiovascular parameters. Apart from the importance of research, it is important for the clinical ICU physiotherapist to be able to predict and recognise haemodynamic instability (HDI) before and during intervention, and to be aware of the potential for the persistence of HDI when intervention has ceased.

Haemodynamic instability is defined as an event which may threaten or produce ischaemia of tissue. Physiotherapists may potentially cause HDI by precipitating an alteration in blood pressure, heart rate, oxygen consumption (VO_2), preload, afterload, myocardial oxygen demand (MVO_2), catecholamine release, intracranial pressure, or arterial blood gases. Whether or not these alterations proceed to changes in cerebral perfusion pressure (CPP), cardiac output (CO), myocardial ischaemia or oxygen tissue delivery (DO_2) depends on the actual technique(s) used, the specific condition and underlying physiology of the patient, and the compensation mechanisms that occur in response to this threat.

Physiotherapy treatment and techniques

Gauging the true effect of physiotherapy intervention on haemodynamic stability in the ventilated patient is made difficult by the fact that many studies do not specify the techniques used or length of treatment. Most studies use the all encompassing term chest physiotherapy. An attempt will be made to look at these techniques as a separate event, as well as studies which combine various treatment methods.

Positioning

Appropriate positioning in ventilated patients can be one of the most useful modalities in facilitating peripheral secretion flow and maximising oxygenation (Kigin 1988). However reservations are often expressed by physiotherapists about the use of extreme positions in the intensive care situation eg head down tilt (HDT) and prone lying.

Interestingly, studies performed on ICU patients by medical staff aim to investigate whether HDT improves haemodynamics. If the patient suffers acute hypotension, thought to be from hypovolaemia, the HDT is utilised in order to shift fluid from the legs into the intravascular space and increase perfusion (Wong et al 1988).

A number of studies on HDT have

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been performed by aviation and space institutions, usually on young healthy humans. Time periods of HDT examined are up to one hour (Wilkins et al 1950).

The most common effects experienced were increases in oxygen consumption (VO_2), carbon dioxide production (VCO_2), right atrial pressure (RAP), and preload (Katkov 1985, Loeppky et al 1987, Lollgen et al 1984, Pricolo 1986). Some investigators (Blomqvist et al 1983, Kubal et al 1984, Nixon et al 1982, Pricolo et al 1986, Sibbald et al 1979) found increases in stroke volume (SV) or cardiac index (CI) while others did not (Gazenko et al 1980, Jennings et al 1985, Katkov et al 1979). Importantly, no studies reported a decrease in SV or CI in any of the subject populations. Most of these studies, however, involved healthy volunteers as subjects and consideration must be given to the effect of an increased preload or VO_2 on a diseased or failing heart. HDT is often considered to be contraindicated in hypertensive patients, yet Lollgen et al (1984) found no increase in mean arterial blood pressure (MAP) in this group of patients. However, if the hypertensive patient had a hypertrophic heart and was on the upward portion of the Starling pressure curve, an increase in preload could tip the patient into failure.

As HDT increases preload, it could be utilised in hypovolaemic patients during techniques which involve the application of positive pressure eg manual hyperinflation. As the section on manual hyperinflation details, positive pressure can result in a decreased cardiac output during a number of conditions. This positioning may prevent these occurrences.

Tilting acute head injured patients head down has been considered dangerous, but Artru et al (1983) found that even in patients with high intracranial pressure (ICP), tilting did not adversely affect haemodynamic or intracerebral parameters.

Despite difficulties with endotracheal tubes and invasive lines, prone positioning is recommended. Phiel and Brown (1976) demonstrated an improvement in PaO_2 in prone, in the patient with acute respiratory failure.

Unilateral positioning

Severe unilateral lung disease can result in dramatic haemodynamic alterations. These include increased pulmonary vascular resistance (PVR), right ventricular failure, decreased cardiac output (CO) and irreversible haemodynamic failure (Crimi et al 1987). Correctly positioning a patient with unilateral lung disease, ie with diseased lung uppermost, can prevent these adverse effects, reduce the interpulmonary functional gradient and the PVR and increase the PaO_2 (Crimi et al 1987, Rivaro et al 1984) Pain and discomfort during repositioning contributes to increased energy expenditure and this can be prevented by analgesics or sedatives (Swinamer et al 1987).

Active and passive exercises

Both active and passive exercises are an important part of intensive care management. They are used to decrease circulatory risks, increase ventilation and improve musculoskeletal function.

Passive exercises in ventilated intensive care patients have been shown to increase metabolic requirements and heart rate by 20 per cent and 10 per cent respectively above resting values (Weissman et al 1984). If the limb has been immobile for only short periods of time or has recently had unknown trauma, pain and the stress response could result. Consideration should be given to communication and gentle handling, beginning with small range movements. Passive movements in non-curarised head injured patients with tonal changes are especially likely to produce changes in HR, ventilation, blood pressure (BP) and metabolism.

Active exercises produce the usual effects of exercise (Hammond and Frolicher 1985), ie an increase in myocardial oxygen consumption (MVO_2), BP, HR, and peripheral dilatation, all of which may place too much stress on an ischaemic heart. It is essential to check the ECG, recent medications, subjective feelings of chest pain, BP and HR prior to active exercise.

Suction

Suctioning of the patient whether by the endotracheal, nasopharyngeal, tracheotomy or oral route, has inherent hazards. There are many well documented occurrences of HDI eg dysrythmias, hypotension, cardiac arrest (Barnes and Kirchoff 1986, Cameron and McMichan 1984, Nuno and Baun 1983, Rosen and Hillard 1960, Shim et al 1969, Walsh et al 1989) and other effects such as hypoxaemia, distress and laryngospasm which may result in increased preload, increased myocardial demand, or cardiac ischaemia (Schwartz et al 1987).

Various methods have been suggested to minimise the oxygen drop during the application of suction, including an oxygen insufflation catheter, (Bodai et al 1987, Guthrie et al 1987, Languher et al 1980) along with hyperoxygenation, hyperinflation, and/or hyperventilation (Baun and Flones 1984) or preventing a disconnection from the ventilator (Brown et al 1983, Cameron and McMichan 1984). Young (1984) suggests that if enough attention is paid to duration, size of catheter, amount of negative pressure, and some method of preoxygenation, arrhythmias will not result.

However, arrhythmias and cardiac arrest during suctioning procedures are well documented despite adequate oxygenation. Rosen and Hillard (1960) indicate that this may be due to an increased venous return on a diseased heart. There is also the possibility of vagal stimulation especially during nasotracheal suction. If sympathetic reflexes are absent, such as may occur in the patient with Guillain-Barré syndrome, bradycardia may result during suctioning (Wilkins 1988). Walsh et al (1987) found that venous oxygen saturation (SvO_2) fell, chiefly due to an increased oxygen consumption (VO_2).

HR also rose in all patients.

Preventing disconnection from the ventilator appears to be an important factor in preventing haemodynamic events. Cameron and McMicham (1984) found that during suction where the ventilator was disconnected, sinus bradycardia, multiple ventricular ectopic beats or tachycardia occurred, but when patients were left connected, no haemodynamic changes resulted. Schwartz et al (1987) claimed that discontinuance of ventilation without suctioning resulted in a rise in left ventricular end diastolic pressure with an increase in cardiac output (CO). The Trach care system (Wilkins 1988), a totally enclosed system where the patient is not disconnected for suctioning, could produce less adverse effects.

Despite the advances in minimising hypoxaemia, suction still appears to be a dangerous procedure. The increase in left ventricular end diastolic pressure, VO_2, HR and catecholamines could all result in ischaemia or arrest in a patient with a diseased heart. Consideration should be given to the plea by Schwartz et al (1987) and Walsh et al (1989) to minimise the use of the procedure, administer sedation and communicate with and provide reassurance to the patient.

Percussion and combined studies

Although chest wall percussion has been re-evaluated (Sutton 1988) it has been shown (Radford et al 1982) to be a valuable technique in loosening secretions. It is often regarded as potentiating HDI, but very few studies have looked at this component alone. On populations consisting solely of ventilated head injured patients, Brimoulle et al (1988), Imle et al (1988) and Paratz and Burns (1992) found that percussion did not significantly increase arterial blood pressure or intracranial pressure (ICP) and that cerebral perfusion pressure (CPP) was well maintained.

Studies combining techniques, usually postural drainage, percussion, vibration and suction, have assessed haemodynamic parameters. Respiratory physiotherapy in mechanically ventilated, critically ill patients has been shown to lower the arterial oxygen (PaO_2) which may precipitate cardiac arrhythmias (Brock-Utne et al 1975). If cardiac output is also decreased, oxygen tissue delivery (DO_2) will also be critically affected. The results of various studies have differed, some showing a decrease in PaO_2 (Connors et al 1980, Huseby et al 1976) some variable responses, (Sykes et al 1970) while others show that PaO_2 is well maintained (Holody and Goldberg 1981, MacKenzie and Shin 1985).

Weissman et al (1984) and Klein et al (1988) examined the responses of critically ill, mechanically ventilated patients, during respiratory physiotherapy usually consisting of positioning the patient in side-lying, percussion, vibration and suction. Heart rate, rate pressure product (RPP), [rate pressure product = systolic arterial pressure x heart rate], VO_2 and VCO_2 were shown to rise significantly in these studies. This could conceivably reduce coronary blood flow, increase myocardial work and cause cardiac ischaemia. Klein et al (1988) found that RPP rose above 15,000, an effect which has been claimed to cause myocardial ischaemia in patients with coronary artery disease (CAD) (Gobel et al 1978), and that fentanyl alleviated the response. In addition to the these parameters, Aitkenhead et al (1984), in adding manual hyperinflation to the treament regime, also found that RPP increased above 15,000 and that there was an increase in catecholamines with physiotherapy. Winthers and Rein (1990) commented that an abrupt surge of catecholamines can be suspected as a potential cause of platelet aggregation and subsequent myocardial infarction.

With the use of real time ST segment analysis, ECG changes may be able to be viewed more carefully. Matthews and Weissman (1988) reported that a significant number of patients with both no history and known CAD experienced ST depression during physiotherapy. These authors comment that respiratory physiotherapy (RPT) appeared to be a particularly inciting event and two of the 11 patients in their study had ST depression each time they underwent RPT.

MacKenzie and Shin (1985) found no significant changes in HR, cardiac index (CI), left ventricular stroke work index, right ventricular stroke work index or intrapulmonary shunt function (Qs/Qt) during RPT. Any alterations noted returned to baseline values within 15 minutes. This was the case even when the treatment lasted up to 67 minutes.

Studies involving percussion are difficult to evaluate due to lack of specificity of techniques. Combined techniques certainly appear able to affect the metabolic requirements of the heart. It is difficult to know whether it is the repositioning, percussion or suction which causes these changes, but it is apparent that fentanyl or similar medications can alleviate the response (Klein et al 1988).

Manual hyperinflation

Manual hyperinflation or bag squeezing is a technique in which an increased tidal volume and oxygen percentage is administered to the patient via an anaesthetic bag. The optimal pattern is long inspiration with an inspiratory plateau, to assist collateral ventilation and stabilisation of alveoli, and a short expiration in order to maximise mean expiratory flow rate (Marini 1984, Scholten et al 1985). Theoretically, adverse haemodynamic effects could result from this ventilation pattern. Consideration should also be given to the haemodynamic effects of providing 100 per cent oxygen during manual hyperinflation as even in normal resting man, it has been shown to produce a decreased cardiac output and increased peripheral resistance (Bryan-Brown 1988).

When mechanical ventilation is applied, consideration is given to the effects of positive pressure on haemodynamics. A ventilatory pattern

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of short inspiration, long expiration, no plateau and decreased airway pressure is recommended.

Before interpreting the few studies of manual hyperinflation on haemodynamics it is necessary to appreciate the effects of positive pressure on haemodynamics. These were well documented in early literature, when mechanical ventilation was first instituted (Blackburn et al 1973, Price 1954). Pressure changes in the thorax can affect cardiac performance, through the thoracic pump mechanism as well as interference with pulmonary blood flow (Hillman 1986, Mushin et al 1980).

Apart from this mechanical impediment to venous return, there appear to be a number of vagal afferents present in the lung which affect cardiovascular responses to positive pressure (Cassidy et al 1986). It has been shown (Cassidy et al 1986) that if the lung is isolated, pressures in the region of 10-30mmHg will cause reflex bradycardia via increased cholinergic - pulmonary C fibres.

This decrease in CO due to both the mechanical effects and stretch receptors is offset by a sympathetic compensatory response from the arterial baroreceptors (Conway 1976). This restoration only results if the sympathetic system is capable of such a response (Rouby 1987). A number of conditions may result in a drop in CO during manual hyperinflation.

A great deal of work has been done on the normal responses to mechanical ventilation (Blackburn et al 1973, Price 1954, Robotham et al 1983). The normal response of BP and HR to increased positive pressure is as follows: immediately after the onset of positive pressure there is a slight rise in BP followed by a decrease in pulse pressure (systolic arterial blood pressure (SAP) minus diastolic arterial blood pressure). With continued pressure there is further narrowing of the pulse pressure and with the release in ventilation a further slight fall and then an overshoot in SAP. If the overshoot is not equal to or greater than 10mmHg, compensatory reflexes are not effective. With respect to HR, an increase appears to occur first and then a decrease on cessation of positive pressure. An abnormal response (demonstrating that compensatory reflexes are not present) would be a continuous decrease in SAP and a slow recovery towards normal, with a decrease in HR (Conway 1976).

A number of factors may be present which will prevent the sympathetic response from working (Hillman 1986, Mushin et al 1980). These include hypovolaemia, (either absolutely or due to peripheral dilatation); any factors which cause increased sympathetic activity in the prestimulus period eg fear, cold, phaeochromcytoma; sympathetic blockade by ganglion blockers, beta blockers or anaesthesia; hypocarbia; a head-up tilt; spinal injury or Guillain-Barré syndrome (Eckenhoff 1963).

There are very few studies of manual hyperinflation reported. Laws and McIntyre (1969) examined the haemodynamic effects of chest physiotherapy (this being defined as manual hyperinflation with expiratory vibrations in the supine and each lateral position). Only six patients were measured. In patients who were unconscious or too ill to resist, the cardiac output (CO) fell by as much as 50 per cent. In the patients who actively resisted, CO rose by figures of up to 50 per cent. Manual hyperinflation pressures were reported to be between 60-100mmHg.

Henman and Guthrie (1983) found a decrease in MAP with three hyperinflation breaths delivered by a ventilator. This effect was magnified if the patient was vasodilated and hypovolaemic. If the inspiratory:expiratory ratio was high, this also caused a decrease in MAP.

On applying manual hyperinflation to head injury patients, Paratz and Burns (1992) found that manual hyperinflation alone increased ICP, end tidal carbon dioxide, MAP and systolic arterial pressure (SAP) without affecting CPP. In head injured patients these responses may have been due to cerebral adjustments, that is the SAP was increasing to compensate for the rise in ICP, and so maintain CPP.

As MHI involves delivering a larger tidal volume, with a longer inspiratory time and shorter expiratory time than mechanical ventilation, there must be more interference with venous return. It is assumed that the sympathetic system rests at a higher point, however there is no normative data which details BP or HR responses. Any studies which attempt to claim that MHI does not affect haemodynamic stability because no change occurred in BP may be describing a lack of a sympathetic response.

Cournand (1948) cautioned that coronary blood flow (CBF) can drop during positive pressure if the inspiratory phase is equal to or longer than expiration. As the heart only receives its blood during diastole, the accompanying tachycardia may decrease CBF (Bryan-Brown 1988).

Mushin et al (1980) are critical of any ventilation pattern that includes an inspiratory plateau. These authors suggest that the resulting decrease in CO will override any improvement in PaO_2, and the result will be a decreased delivery of oxygen to the tissues (DO_2).

The timing methods and types of circuits used during MHI differ between hospitals, making it difficult to gather normative data. The physiotherapist, however, needs to be able to recognize HDI during or prior to treatment. Chernow and Steigman (1988) and Szold et al (1989) describe how systolic arterial pressure variation may be used as as a test prior to treatment to predict hypovolaemia and possible haemodynamic instability. If a head-up tilt results in a decrease in systolic blood pressure greater than 20mmHg or a heart rate increase greater than 20 beats per minute, vascular volume depletion is present. If any of the other conditions which prevent a sympathetic response from working are present, cardiac output may drop during MHI.

In summary, the patient may experience haemodynamic instability if a number of conditions are present. If a patient is unable to tolerate a decrease in preload or in coronary blood flow,

they may not tolerate manual hyperinflation.

Recommendations

While it is important to attempt to predict HDI, it should be appreciated that 78 per cent of intensive care patients are likely to experience arrhythmias spontaneously (Artucio and Pereira 1990). Shoemaker et al (1989) noted 637 unanticipated monitored events in 250 patients. One of the most important findings was that of Bergbon-Erborg and Haljamame (1989) who found anxiety, fear and the inability to communicate rated higher than suctioning as perceived causes of distress in people who had experienced intensive care treatment. Sedation and/or analgesia have been shown to alleviate the adverse response to physiotherapy in some studies (Klein et al 1988) and should perhaps be used together with increased explanation and reassurance.

The present state of research in haemodynamic stability during respiratory physiotherapy in intensive care patients is inconclusive. As treatment techniques may vary in their effect according to the actual pathophysiology of the patient, studies which use a heterogenous group of patients are unsatisfactory. There is a need for widespread multi-centre documentation of haemodymanic events during physiotherapy. This would indicate if there were correlations betweeen techniques, conditions, and haemodynamic parameters. Normal data on the responses of heart rate and blood pressure to manual hyperinflation is also necessary in order to interpret the results of further clinical trials. Single case studies linking the results with the pathophysiology of the presenting conditions would add to the present state of knowledge. It is apppreciated that the patients with most invasive monitoring are usually very unstable and often not suitable for inclusion in a controlled trial, but for this same reason are better suited to a single case study.

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